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## CASE REPORT

***Streptococcus anginosus* Lung Infection and Empyema: A Case Report and Review of the Literature**Nishita Tripathi<sup>1\*</sup>, MD; Kuldeep Ghosh<sup>2</sup>, MD; Anupama Raghuram<sup>1</sup>, MD<sup>1</sup>Division of Infectious Diseases, Department of Medicine, School of Medicine, University of Louisville, Louisville, KY, USA; <sup>2</sup>Division of Internal Medicine, Metropolitan Hospital, New York Medical College, New York City, NY, USA

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We report a patient with empyema in the setting of an unresolved *Streptococcus anginosus* pneumonia for over a month after two courses of antimicrobial treatment. A chest computed tomography (CT) revealed a localized pleural fluid collection. The pleural fluid cultures grew *S. anginosus* resistant to clindamycin and erythromycin. The patient was treated with ampicillin-sulbactam plus surgical interventions. Initially, the patient underwent CT-guided placement of chest tube

with instillation of tissue plasminogen activator. Subsequently, he required a video-assisted thoracoscopy with decortication and drainage of empyema, followed by a remarkable improvement in his overall condition. Our case highlights the infections caused by the *Streptococcus milleri* group (SMG) in individuals with an unresolved pneumonia. Such patients should be diagnosed accurately and treated aggressively with rapid and effective interventions.

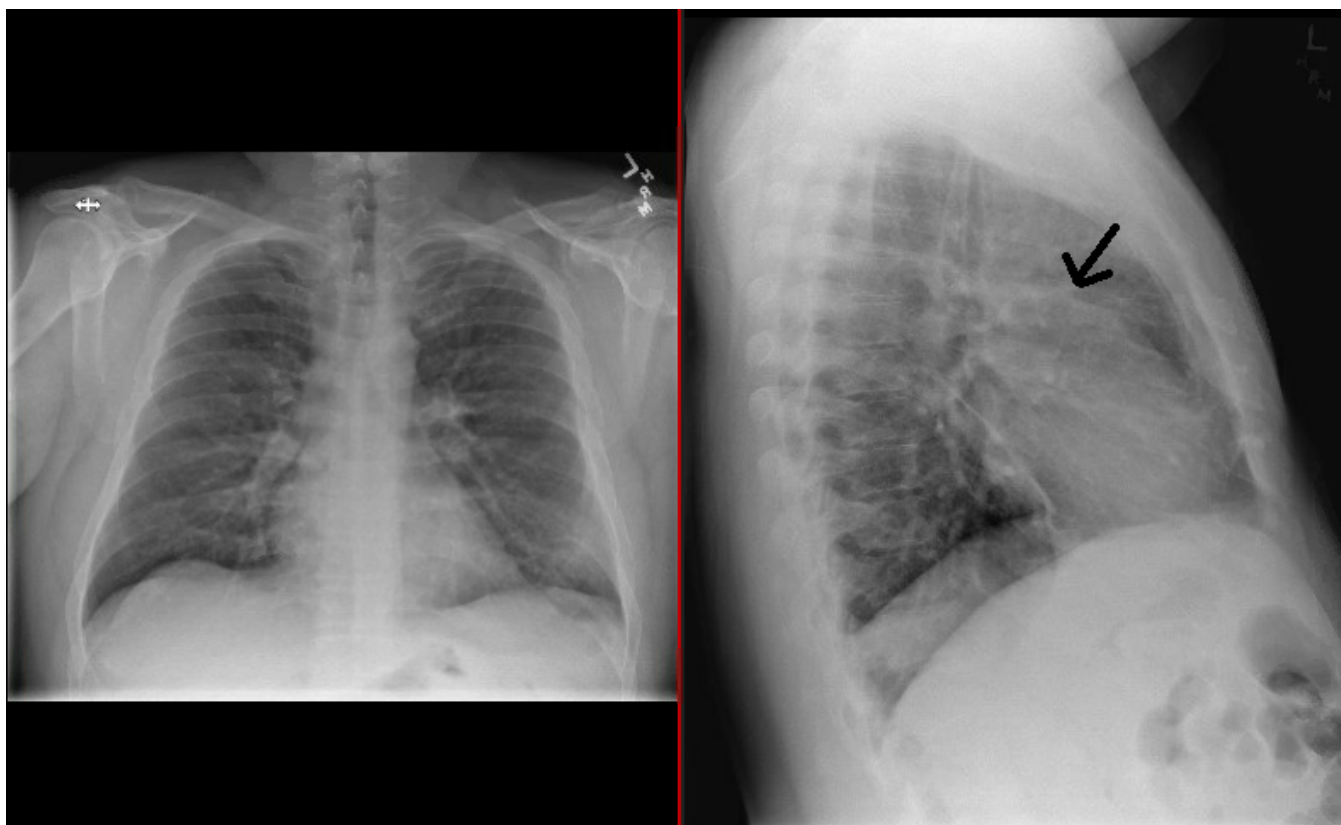
**Introduction**

*Streptococci milleri* group (SMG) have been known to cause purulent infections.[1] They are commensal organisms found in the mucosa lining the oral cavity, oropharynx, gastrointestinal tracts and genitourinary tracts in both adults and children.[2] These organisms have been known to cause periodontal abscess.[2, 3] They are commonly associated with both monomicrobial and polymicrobial infections. Monomicrobial infections are more common in head and neck infections while gastrointestinal infections are polymicrobial in nature involving anaerobes.[2, 4] Anaerobic organisms found in the polymicrobial infection have been reported to enhance SMG infections.[4–7] *Streptococcus anginosus* is strongly associated with infections of the skin and soft tissue, as well as gastrointestinal and genitourinary infections.[6] These organisms are unique in their ability to form abscess, which differentiates them from *S. pyogenes* and *S. agalactiae*. [3] Most often, these organisms have been implicated in causing parapneumonic effusions and abscess.[8, 9] Identification of these organisms is essential for timely diagnosis, and aggressive treatment is required. We describe a case of empyema caused by SMG in a patient with an unresolved left-sided pneumonia.

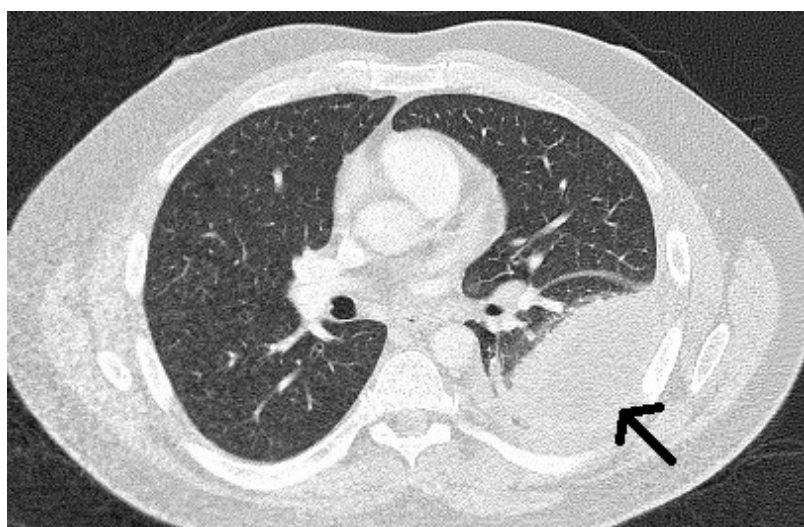
**Case Report**

A 37-year-old Caucasian male was evaluated in the emergency room (ER) for 4–6 weeks of cough, white sputum, shortness of breath, and weight loss. A month prior to his current presentation, he was diagnosed with radiologically confirmed left lower lobar pneumonia (**Figure 1**) and was treated with a 4-day course of 250 mg of azithromycin as an outpatient. A month after the initial presentation, he reported similar complaints and was prescribed 100 mg twice a day of doxycycline for 28 days. The patient has worked as a gravedigger for 14 years. He smoked one pack of cigarettes per day over the last 20 years and had no history of alcohol abuse. His medical history was significant for chronic bronchitis and adenomatous colonic polyps diagnosed in the past year.

On physical examination, he had decreased breath sounds in the left middle and lower lung fields. Initial workup revealed a white blood cell count (WBC) of 18,210 cells/mm<sup>3</sup>. A chest computed tomography (CT) showed a left-sided pleural effusion and compressive atelectasis of the left lung, consistent with empyema (**Figure 2**). He was admitted to the hospital for further management. He underwent CT-guided place-



**Figure 1.** Chest X-ray done prior to hospital admission showed left lower lobe pneumonia.



**Figure 2.** Computerized tomography of the chest at hospital admission shows left base pleural collection suggestive of empyema.

ment of pleural catheter with tissue plasminogen activator (TPA) irrigation for the drainage of pleural fluid during the first 3 days of his admission. A follow-up chest X-ray at day 3 of hospitalization showed a reduction in the fluid collection with persistence of left-sided complicated effusion. A panoramic mandibular X-ray was performed due to his poor dentition, showing no findings of periapical abscess. The microbiological cultures of blood and sputum were negative; however, at day 4 of his admission, pleural fluid cultures grew *S. anginosus*, which was resistant to clindamycin and erythromycin (Table 1). Due to the presence of significant fluid on subsequent radiological studies, he underwent video-Assisted thoracoscopic surgery (VATS) with lateral decortication and drainage of empyema with placement of a chest tube on day 7 of the hospitalization. A subsequent CT showed improved atelectasis and consolidation in the left middle and lower lobes (Figure 3). The pleural fluid cytology and histopathological analysis excluded malignancy. The patient was started on intravenous (IV) ampicillin-sulbactam 3 mg every 6 hours from day 2 of hospitalization until day 12. He was switched to 500 mg of oral amoxicillin during the last two days of his hospitalization and was prescribed the same for an additional five days after being discharged. The patient improved clinically and was discharged on day 14 of hospitalization. The patient continued to show remarkable marked improvement during subsequent outpatient follow-up appointments. A radiological assessment three months after his admission to the hospital showed complete resolution of the left-sided empyema (Figure 4).

## Discussion

We herein present a case of pleural empyema caused by *S. anginosus* in a young patient with an unresolved pneumonia. Since the SMG are not the most common pathogens known to cause empyema in the United States, it is important to suspect them in patients who do not improve on empiric antibiotic therapy.[10]

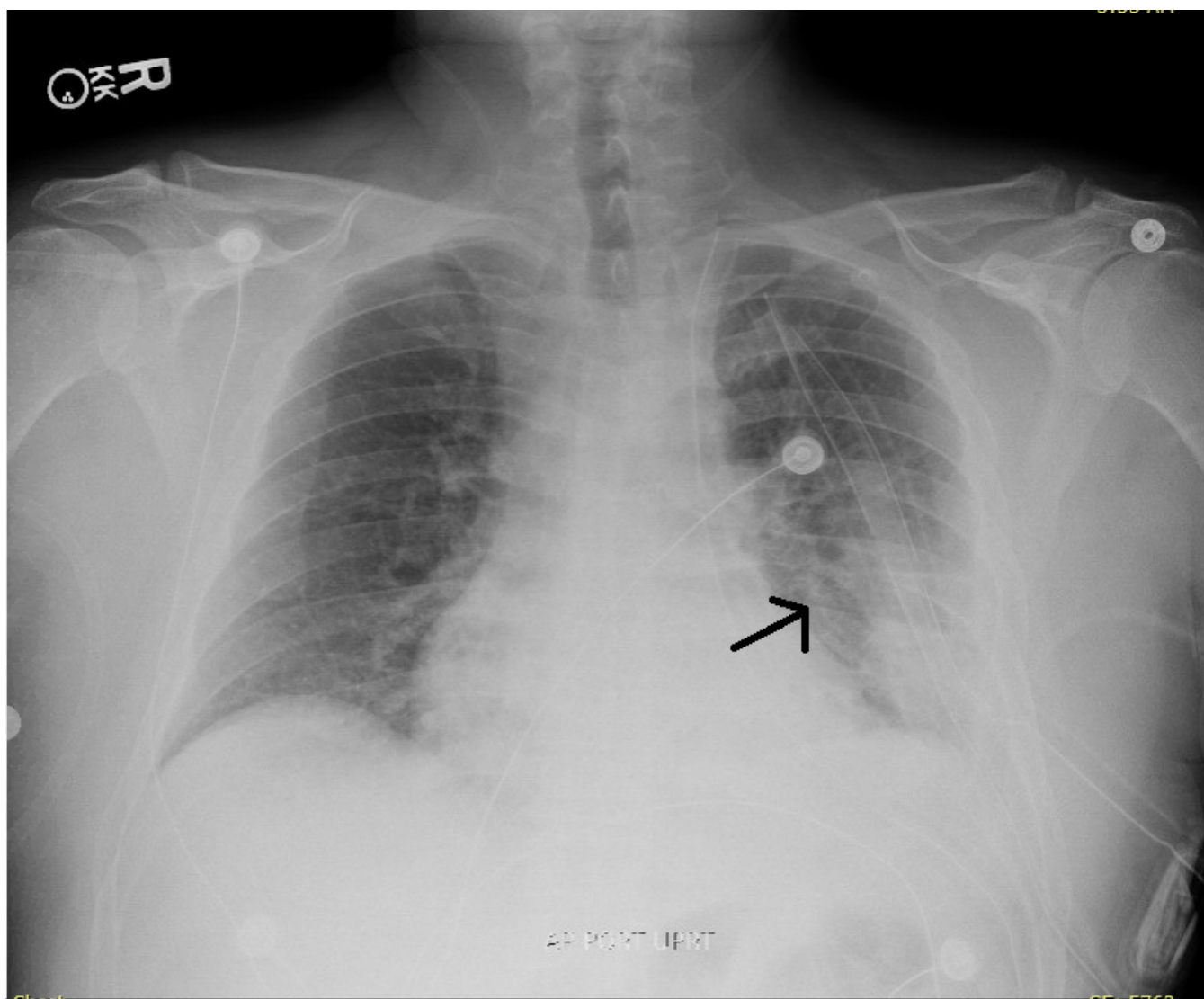
Pleural empyema is a bacterial infection resulting in the formation of pus in the pleural cavity.[10] It is an uncommon complication of community-acquired pneumonia (CAP), with approximately 32,000 cases per year in the United States. Around 20 percent of patients with pneumonia develop a parapneumonic effusion, which may lead to empyema.[11] The mean time for the development of empyema has been reported as 18 days.[8, 12] Empyema can be due to untreated pneumonia. Accumulation of frank pus and microbes in the pleural cavity leads to the development of empyema.[13] The pleural infection can be divided into three stages. Stage 1 is the exudative phase, which is characterized by the accumulation of clear fluid. This is a simple parapneumonic effusion due to the inflammation caused by the pneumonia. During Stage 2, the sim-

ple effusion may undergo fibrinopurulent deposition, triggered by bacterial invasion leading to the formation of empyema. At Stage 3, there is a widespread scarring and pleural peeling, causing restriction of lung re-expansion. Increasing prevalence of empyema caused by SMG bacteria should raise concern among clinicians. The predisposing factors for SMG empyema include old age (median ages 60-68), mucosal disturbance (e.g., sinusitis, periodontal abscess), excessive alcohol consumption, diabetes mellitus, previous surgical procedures involving instrumentation in the thorax, enteric diseases (e.g., esophageal perforation) and immunosuppressive states, such as malignancy.[4, 5, 9, 14] Cases of pleural empyema have also been described in young patients with none of these risk factors.[10] The patient in our study was young and had non-resolving pneumonia as a risk factor. A case series of 25 patients found that pneumonia was a predisposing factor in around 40 percent of patients who developed SMG empyema, thereby stressing the role of these organisms in pulmonary infections.[8] According to another study, pleural effusion was reported in 54.5 percent of patients with SMG infections. The study also found that pleural effusion was more commonly associated with SMG infection than infections caused by other pathogens, including but not limited to *S. pneumoniae*. [15] Our patient also initially presented with a loculated empyema, which was consistent with previously reported cases. Although our patient had poor dentition on an oral examination, he did not have any periodontal abscess, which was unique since dental abscess was found to be associated with 40 percent of patients with SMG empyema.[4] Therefore, thorough oral examination should be conducted in suspected cases. Since SMG are not commonly considered pathogens when evaluating patients for sputum culture, it is often difficult to distinguish them from contaminants. Therefore, physicians must assess the patients who have a positive sputum bacterial culture carefully.

SMG empyema should be treated aggressively with antimicrobial drugs in combination with surgical interventions. The antimicrobial therapy may be guided by susceptibility studies. SMG are generally susceptible to beta-lactam agents. Commonly used agents include penicillin, ampicillin, and cephalosporin. For those allergic to penicillin, erythromycin, clindamycin or vancomycin could be used as alternatives.[4, 5] However, cases of resistance to erythromycin and clindamycin have been reported, consistent with our case report.[16] The antibiotics showing 100 percent susceptibility are vancomycin, imipenem and teicoplanin.[13] Amoxicillin-clavulanic acid can be used in cases of polymicrobial infection.[3] Our patient was treated with IV ampicillin-sulbactam during the course of his hospitalization and was prescribed amoxicillin-clavulanic acid at the time of discharge to ensure adequate coverage. Inadequate treatment with antimicrobials during the acute phase leads to the progression

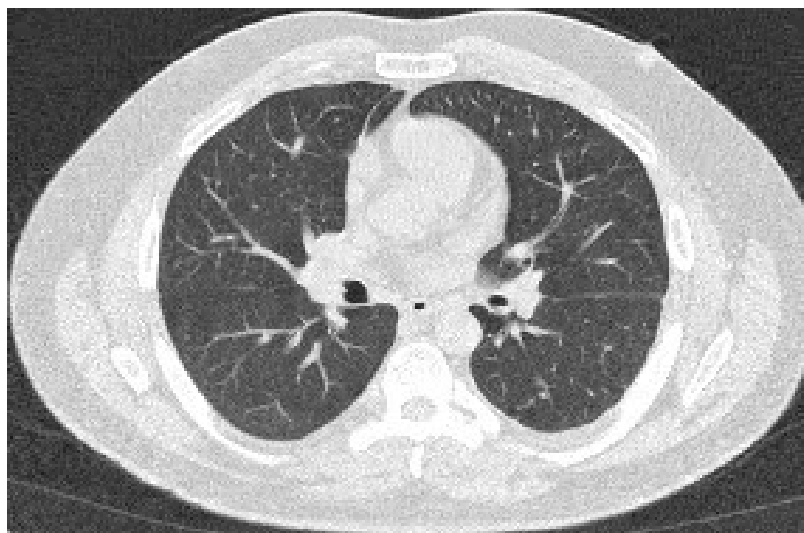
**Table 1.** Table showing antibiotic susceptibility of *Streptococcus anginosus* isolated in the pleural fluid culture at day 4 of hospital admission.

Antibiotic	Interpretation
Ceftriaxone	Susceptible
Clindamycin	Resistant
Erythromycin	Resistant
Levofloxacin	Susceptible
Pencillin G	Susceptible
Vancomycin	Susceptible



**Figure 3.** Follow-up chest X-ray imaging done post-VATS on day 7 of hospitalization showed reduction in size of opacity in left hemithorax.





**Figure 4.** Follow-up computerized tomography of the chest 3 months after hospital admission showed complete resolution of left-sided empyema.

of empyema.[17] The duration of antimicrobial therapy may be variable; however, a minimum of 2 weeks of antibacterial therapy from the time of drainage and defervescence is recommended.[18] Antimicrobial treatment alone is not recommended for empyema since there is a wide fluctuation in antibiotic penetration into the abscesses, independent of the dosage given.[8, 9] Empyema often requires drainage.[13] Intrapleural instillation of streptokinase enhances the clearance of pleural cavity and could be used in the management of effusions and empyema.[4] Complete drainage of empyema in the acute phase is important in achieving re-expansion of atelectasis and obtaining a satisfactory outcome.[17] The majority of the SMG empyema require surgical intervention for definitive therapy. Hence, they should be considered for early operative intervention due to the unrelenting nature of the infection. Early surgical intervention is associated with shorter duration of hospitalization, decreased mortality [1] and favorable outcomes in patients with SMG respiratory infection. Thoracocentesis is useful in the management of uncomplicated pleural effusion (Stage 1) and helps in the diagnosis of complicated effusion. VATS decortication is a feasible and effective procedure for the treatment of empyema refractory to conventional therapy.[19] VATS is the first-line approach in all patients with Stage 2 or Stage 3 empyema who can tolerate single-lung ventilation. However, thoracotomy is recommended for patients who may not be able to tolerate single-lung ventilation or continue to have recurrence. Patients with chronic empyema are treated through surgical interventions, as opposed

to pleural drainage procedures, since complete evacuation of pleural space followed by obliteration of empyema plays a key role in preventing recurrence. These patients may be treated with VATS or open thoracotomy. Open thoracic window and marsupialization of infected thoracic cavity with resection of several ribs and dressing changes is performed in patients who are medically unfit to tolerate VATS or decortication procedures.[18] Despite undergoing pleural drainage interventions during the initial course of his hospitalization, our patient underwent VATS subsequently due to the recurrence of fluid collection, as evident in radiological assessments. Delay in diagnosis, failure to institute appropriate antimicrobial therapy and inadequate drainage contribute to increased morbidity and mortality.[13, 17] Aggressive management with rapid and effective intervention can help to reduce in-hospital mortality, morbidity, and duration of hospital stay and tends to result in overall good long-term outcomes.[13, 17]

## Conclusion

In conclusion, *S. anginosus* empyema should be suspected in any patient with unresolved respiratory infection not responding to empirical antibiotic therapies, consolidation on repeated radiological imaging and a possible history of odontogenic infection. Taking a careful history, performing a thorough physical exam, and managing the infection aggressively with appropriate antibiotics, drainage and, if required, surgical debridement is essential to ensure resolution.

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